```
**AKI and CKD and ESRD**
```

- **Flank Mass**
- Hydronephrosis
- RCC
- Renal cyst
- ADPKD
- **Bilateral Flank mass**
- ADPKD
- Bilateral hydronephrosis
- Diabetic Nephropathy in early days
- Amyloidosis

  ```
  CKD with enlarged kidney
  ```

- DM
- HIV
- ADPKD
- MYELOMA
- AMYLOIDOSIS
- **how to differentiate AKI from CKD**
- check creat and compare with previous record
- if not available, repeat after 2 months
- normal output favours ckd
- normal pth favours aki
- Anemia favours CKD

- **how to differentiate Pre-renal from Renal azotemia**
- Fractional excretion of sodium is less than 1% in pre renal, more than 4 in renal
- BUN to creat ratio is more than 20 in pre renal, less than 10 in renal
- **pregnancy**
- if creat less than 1.4.. not high risk
- if more than 1.4 - during pregnancy pt is at risk of disease progression and fetal loss

Renal biopsy method

- – – (Non Diabetic Glomerular disease, Unusal AKI, Unusual proteinuria)
- admit 1 day ago
- bleeding profile
- serial bp recordings
- – prone position, localize with usg, measure distance and depth, then take sample with gun
- – 2 samples taken (1 in normal saline, 1 in formaldehyde)
- complications - trauma, infection, bleed
- minor bleed, perinephric, is seen in more than 50%
- Contraindications - sepsis, bleeding disorder, BP above 190, solitary kidney, horse shoe kidney

Cardiorenal Syndrome

- Deterioration of one organ will effect other. 5 recognized patterns

1. AKI due to CCF
2. CKD due to CCF
3. Acute heart failure due to CKD
4. Decompensation of chronic heart failure due to CKD
5. Cardiac and renal deterioration due to some other cause - sepsis etc

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ADPKD

- Asymptomatic
- Discomfort
- Infection
- Bleed
- Hypertension
- Renal failure
- CVA
- Polycythemia

association

- Cystic liver, spleen, brain, lungs
- Berry aneurysm
- MVP

RCC

- Asymptomatic
- triad of (Painless hematuria, Loin heaviness, Mass)
- others. (PUO, Hypertension, Anemia, polycythemia,nMetastasis)
- – *TREATMENT*
- Surgery
- RFA
- TK inhibit (Sorafenib, sunitinib)
- temsirolimus
- Bevacizumab (monoclonal anti VEGF)

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Myeloma Reneal Injury
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1. Cast Nephropathy
2. Tubular blockage
3. GN (MGN, MPGN)
4. Recurrent infection (Pyelonephritis)
5. Infiltration by Plasma cells
6. light chain disease
7. Amyloidosis
8. Renal vein thrombosis

```
Amyloidosis
```

- Light Chain, Heavy chain
- Hereditary, systemic
- work up - rectal or subcut fat biopsy or organ biopsy, imunocytology studies
- treatment of amyloidosis
- Hereditary - immunotherapy like in myeloma and bone marrow transplant
- prognosis is poor – renal - 3 years cardiac - 2 years (Famous patient - Pervez Musharraf)

CRYOGLOBULINEMIA

- Immunoglobulin that **REVERSIBLY** precipitate in low temperature
- – **Types**

1. I - associated with gammopathy (myeloma, MGUS, waldenstorm)
2. II - associated with rheumatological diseases (its an IgM against IgG)
3. III - both IgM and IgG

Some point to Remember

- I, II are monoclonal, III is polyclonal
- II,III are called mixed (10% develop CKD after 10-15 years)
- I and II are like systemic Vasculitis (Rheumatological diseases, lymphoproliferative, hepatic, HCV)
- when no cause found, its called essential
- Renal involvement is seen in II.

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FEATURES
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- purpura
- urticaria
- Arthralgia
- weakness
- skin biopsy - leukocytoclastic

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RENAL INVOLVEMENT
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- Asymptomatic proteinuria with hematuria (RFTs mostly normal)
- Nephritic in 25%
- Nephrotic in 5%
- Anuric or oliguric failure

```
INVESTIGATIONS
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- Cryoglobulin (IgM or IgG, with kappa or lambda)
- Significantly low C4
- mildly low C3
- renal biopsy – **MPGN, or intraluminal thrombi in small vessels**

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TREATMENT
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- If Hep B/C associated ➡️ Treatment of Hep B, or C
- steroids
- +/- Cyclophosphamide
- **ACUTE SEVERE** ➡️ Plasmapheresis

```
AKI vs CKD vs ESRD
```

1. **AKI**

- Renal failure develops in *HOURS to FEW DAYS*
- creat increases 0.3mg in 48 hours

2. **CKD**

- Slow, persistent decline in kidney functions.
- Elevated creatinine for more than 3 months or eGFR less than 60

3. **ESRD**

- when patient needs renal replacement therapy

```
CKD STAGING
```

- Description should cover both components (Staging by GFR + Albuminuria description)

1. GFR above 90

- (with some evidence of kidney damage - RFTs, Proteinuria, Hematuria, USG)
- *__REMEMBER__ – without evidence of kidney damage, its not CKD

2. 60-89

- (with some evidence of kidney damage - RFTs, Proteinuria, Hematuria, USG)
- without evidence of kidney damage, its not CKD

3. (a) - 45-59

4. (b) 30-44

5. 15-29

6. less than 15

- 1 to 3 are usually asymptomatic

- Symptoms usually start from 4 onward

- – **ALBUMINURIA** –

- A1 = ACR below 30

- A2 = ACR 30-299

- A3 = ACR above 300

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NOTES
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- **FeNa** – (Fractional excretion of Sodium)

- Pre renal is less than 1%

- intra-renal is 2%

- post renal is 4%

- (if patient taking diuretics, then FeNa will be falsely high, in these patients calculate *FeUrea* which is not altered by diuretics)

- **BUN/creat ratio**

- Normal is 10-20:1

- Pre Renal is more than 20:1

- Intra-renal is less than 10:1

- Post renal is 10-20:1

- – √ *(serum creatinine used as marker depends upon muscle mass — hence low mass = falsely low creatinine)*

- **Albuminuria**

- – (Albuminuria is subtle indicator of)

- Kidney dysfunction

- Microvascular health

- Endothelial function

```
              INVESTIGATIONS
```

1. CBC
2. RFTs
3. Electrolytes
4. LFTs
5. Urine complete

- Proteins
- FeNa
- PCR , ACR
- Casts (RBC = GN, WBC = pyelonephritis, muddy = ATN, sterile pyuria = AIN)

6. USG
7. Non contrast CT
8. FeNa or FeUrea
9. Kidney Biopsy (when sudden decline in RFTs or when multiple risk factors are present)
10. Old age and no obvious cause - UPEP or SPEP for myeloma (and SFLC, UIFE, SIFE)

```
how will you treat
```

1. educate
2. make an agreement about Rx
3. control of risk factors and avoid nephrotoxic drugs
4. prepare for dialysis
5. make fistula
6. dialysis
7. transplant

- – APPROACH
- – **SLOW THE PROGRESS**
- avoid Nephrotoxic drugs
- BP control
- glycemic control
- dyslipidemia control
- Proteinuria management
- stop smoking
- obesity management
- Dietary (restriction of protein, potassium, phosphate)
- – *Educate, counsel and prepare for RRT*
- – save non dominant arm and proceed to fistula formation.
- – discuss about transplant, Hemodialysis, PD
- – ✓ Very frail , or pt with multiple Comorbidities may like not to go for RRT and for them, Palliative care is started. Symptomatic relief

CAUSE SPECIFIC TREATMENT

- Effort should be made to identify the cause because some causes have good response to treatment (lupus, ANCA, Obstruction, AIN)
- HRS - albumin and terlipressin
- GN - immunosuppressants with steroid and steroid sparing agent
- AIN may also need steroids if not responding to supportive care like withdrawal of drug, adequate hydration etc
- Obstruction will need prostste surgery or lithotripsy.

RENAL REPLACEMENT THERAPY

1. Dialysis

- Hemodialysis (Hemodialysis, Hemofiltration, Hemodiafiltration, CRRT
- Peritoneal

2. Transplant

- Auto, Allo, Iso, Xeno

HEMODIALYSIS

- Semipermeable membrane with dialysate fluid.
- Solutes movement by diffusion (means along the concentration gradient)
- Best for small solute clearance

HEMOFILTRATION

- Semipermeable membrane with water only
- Solutes movement is by convection
- (convection means movement on basis of density, weight, heat)
- Good for medium size solute clearence
- Then replacement fluid is added after blood exits the henofilter.

HEMODIAFILTRATION

- Combination of diffusion and convection

CRRT

- in hemodynamically unstable patients (Like ICU pt) because in these situations there is need of a slower rate of fluid removal
- CRRT causes less hypotension than HD
- These patients require a large volume of fluid (drugs, iv drips) and CRRT can prevent an overloaded state.
- One other situation is pt with brain injury, where HD pose risk of disequilibrium syndrome.
- complications - infection, bleed, thrombosis, removal of AA, micronutrients, water soluble vitamins. causing negative nitrogen balance. calories and protein intake needed

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TRANSPLANT

- *Types*

1. Auto - same person
2. Iso - twins
3. Allo - same species
4. Xeno - different species

- Orthotopic - transplant in anatomical location
- Heterotopic - in different anatomical location
- – 10 yr survival –

1. Cadaveric - 60%
2. Living - 70%

- – *POST OP Problems* –
- ATN
- Vessel thrombosis
- Urine leak
- UTI
- – *REJECTIONS* –

1. Hyperacute (minutes to hours)

- due to preexisting antibodies against HLA 1
- IgG
- Rare, because HLA matching is done before procedure

2. Acute (less than 6m)

- risk greatest in first 3 weeks
- HLA mismatch, CMV infection, Cyclosporine toxicity, graft ATN
- – *CMV is most common viral Infection in solid transplant - give GANCICLOVIR* –

3. Chronic (after 6m)

- both immune and non immune factors
- Fibrosis
- – *(GRAFT VS HOST DISEASE)*
- donor lymphos in susceptible host

4. *(POST TRANSPLANT RECURRENCE)*

- MPGN
- FSGS
- MGN

5. *POST OP EVENTS*

- CMV (fever, flu, IMN like picture - give iv ganciclovir)
- Melanoma with Liver (pain RUQ , high LFTs)

DIALYSIS

PERITONEAL DIALYSIS

- Can be performed at home
- has shown mortality benefits in upto first 3 yeats.
- Contraindications - hernia, adhesions, peritoneal shunts
- Cathetor placed *PERMANENT SILASTIC CATHETOR*- cathetor mature in 2 weeks - pt get trained at dialysis centre for 2 weeks -

1. Automated PD (By machine) - fluid is flown in peritoneum - fluid made to move around - withdrawn after a specific time - *USUALLY NIGHT TIME* but if needed cycle can be repeated several times in a day
2. CAPD (continuous ambulatory PD) - pt remain mobile - 2 L isotonic fluid given in peritoneal cavity and left for 4-6 hrs - this fluid is made hyperosmolar by adding glucose polymers (this make vascular fluid to move in this hyperosmolar fluid across osmolarity gradient)- metabolic waste products move into fluid across peritoneal membrane by concentration gradient - fluid removed - cycle repeated 4 times daily

COMPLICATIONS

1. *PERITONITIS*

- present as abd wall pain, fluid becomes cloudy, systemic sepsis, WBC more than 100/ml *MANAGEMENT* - culture - intraperitoneal Axbx (tobramycin, vancomycin) - sometimes cathetor removal

2. *CATHETOR SITE INFECTION*

- Erythema and pus at exit site - give Axbx or surgical drainage

3. *ULTRAFILTRATION FAILURE*

- due to membrane damage - result in overload - manage by replacing glucose with glucose polymers

4. *MEMBRANE FAILURE*

- by scarring - inadequate urea clearence - manage by increasing exchange volume or using Automated PD or switch to Hemodialysis

5. *ENCAPSULATING PERITONEAL SCLEROSIS*

- long term complication - result in obstruction, malnutrition - manage by switch to Hemodialysis

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HEMODIALYSIS IS BEST METHOD FOR REMOVAL OF SMALL SOLUTES

- initially 1-2 hours session
- initial short duration is to avoid removing excess fluid because excess rapid removal can cause disequilibrium syndrome.
- In subsequent days or weeks, session can be increased to 4-5 hours alternate day or 2-3 hours daily.
- For cathetor - IJV or FV is better than subclavian.
- Subclavian has risk of bleeding. and if stenosis or Thrombosis occurs, it will reduce chance of fistula success if needed in future.
- Patient education and planning should start from stage IV.
- Fistula be prepared upto 1 year before estimated need of dialysis.
- In Fistula , veins will need 4-6 weeks for arterialization
- Waiting for uremic symptoms will add risk, particularly of malnutrition.
- Considered better to *start healthy*.
- study of heart and renal protection (SHARP) having dialysis and non-dialysis requiring CKD patients showed a 17% reduction in cardiovascular death and major cardiovascular events with simvastatin-ezetimibe treatment. Cardioprotective strategies such as beta-blockers, aspirin, and renin-angiotensin-aldosterone system inhibitors are recommended in dialysis patients based on their cardiovascular risk profile.
- intermittent or continuous. Continuous intravascular procedures are preferable in those who are hemodynamically unstable or have significant volume overload.

INDICATION

- AKI
- Enceph
- Pericarditis
- Pulmonary edema
- Hyperkalemia
- Neuropathy
- Intractable git symptoms
- Asymptomatic but GFR below 10
- Toxin removal
- – – √ (Decision should be based upon)
- Symptoms of renal failure
- Volume or bp dysregulation
- Nutrition maintenance
- Cognition

- – not in asymptomatic rft decline. –
- – ✓ *If patient decline HD* –
- Conservative management
- Correction of reversibles
- Diet - adequate calories, with calcium and vit d but protein, salt, potassium and phosphate reduction.
- Pain with analgesia
- Pruritus with low dose pregablin or gabapentin
- Anemia
- psychosocial support

DIALYSATE

- *Temperature* 35-38
- Cold fluid cause hypothermia in unconscious and shivering in conscious
- High, 40 , temperature cause blood protein denaturation
- – *Osmolality* –
- Hyper cause hypernatremia
- Low causes hyponatremia and hyperkalemia
- Bicarb buffered Dialysate is better than acetate buffered.
- Bicarb buffered still contain some acetate that will increase risk of acedemia, hypotension and arrythmia.

IMPLANTABLE ARTIFICIAL KIDNEY

- High efficiency filter
- Hemocartridge
- Biocartridge
- Bioreactor made by harvested tubular cells

VASCULAR ACCESS

- Catheter
- Fistula (as distal as possible)
- Graft
- Tunnel cathetor

SAVE NON DOMINANT ARM

- no trauma
- tight clothes/jewelry
- heavy weight
- sleeping on the arm

- Avoid sampling
- Avoid BP

FLOWS

- Blood 300-500ml/min
- Dialysate 500-800 in counter current
- Dialysate side has negative pressure that pulls in the fluid from blood side
- Urea reduction ratio (fraction of BUN reduced per dialysis) - ideally be 60-70%.
- Home dialysis - 3-6 nights , each for 6-8 hours
- In pregnancy *Long frequent dialysis* is recommended, either home or hospital

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DIALYSIS COMPLICATIONS

1. CVS (hypotension, pericarditis, pulmonary edema, Arrythmias , IHD)
2. CNS (Disequilibrium)
3. Infections (B C HIV)
4. Musculoskeletal (cramps)
5. Vascular line (Air embolism, bleed, infections, stenosis or thrombosis)
6. Others (Hypersensitivity, Hemolysis, Amyloidosis, Malnutrition)

INTRADIALYTIC HYPOTENSION

- Systolic Below 90 is bad.
- increase mortality
- LV wall motion abnormalities (myocardial stunning)
- Dizziness, lightheaded, nausea
- Needs urgent bolus of 100ml saline

ARRYTHMIAS

- MC are VF, PEA and Asystole
- due to Potassium
- due to acidosis
- lead to hypotension, chest pain, syncope, ppt heart failure
- *Check and correct Potassium, Acidosis*
- *Review drugs*

DISEQUILIBRIUM

- neurologic deterioration
- restlessness
- Headache
- confusion
- muscle twitching
- Seizures
- coma
- Typically in first dialysis. Fast ultrafiltration - Blood urea is removed faster but CSF urea decreases slowly – high gradient between CSF and blood urea - water movement into the CNS - increased ICP
- (acceptable rate is urea reduction 40% in 2 hours)

INFECTIONS TRANSMISSION

- B,C, HIV

CRAMPS

- Hypotension
- high ultrafiltration rate
- hypovolemia
- low-sodium Dialysate
- – These cause vasoconstriction and reduce perfusion to muscles

DIALYZER REACTION

- Like anaphylaxis
- (give i.v antihistamines, steroids, and epinephrine)

HEMOLYSIS

- is emergency
- Indicated by
- port wine stain along venous track
- Fall in Hct
- Pink centrifuge of blood

AIR EMBOLISM

- Foam noted in Dialysate port
- Churning sound on chest auscultation
- Place in left lateral recombinant
- Give 100% oxygen by mask

HEMORRHAGE

- due to anticoagulation needed during dialysis
- Dialysis needles get disconnected
- *Stop dialysis*
- *identify source*
- *Heparin free dialysis*
- *– between next dialysis –*
- Pulmonary Edema (increase ultrafiltrate)
- Weight gain (increase ultrafiltrate)
- Infections (vascular access line)

OTHERS

- Arteriosclerosis
- Hypertension, specially in chronic overload patients (give poorly dialysed antihypertensive like carvedilol)
- Amyloidosis (Arthralgias)
- high FGF 23 - AFib, LVF
- contribute to mortality (FGF 23, high phosphate, high calcium, pth abnormalities)

CKD DIET

- consume fresh fruits instead of pre packaged
- Potato, pasta , rice only 3 days/week instead of daily

1. protein*

- stage 1-4 = 0.7gm/kg/day
- On dialysis= 1.2gm/kg/day
- to increase calories without protein increase and without adding CVS risk
- ویجیٹیبل آئل
- شہد چینی شربت

2. Sodium less than 2gm
3. Calcium max 1200 *(on dialysis 1200-2000)*
4. Potassium, phosphate adjusted according to Lab *on dialysis potassium 2000-3000, Phosphate 800*
5. fluid restriction not needed if urine output is adequate *on dialysis = 1000 + urine output* fluid restriction not needed if urine output is adequate *on dialysis = 1000 + urine output*
6. Vit D (lab testing should be 25-oh vit d , not 1-25 dihydroxy

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COMPLICATIONS

- all patients needs to be monitored for

1. Anemia
2. Electrolytes and ACIDOSIS
3. Bone Disease
4. HTN
5. Fluid
6. GIT
7. Cardiac
8. Resp
9. CNS
10. ENDO

ANEMIA

1. Causes of Anemia

- EPO Deficiency
- GIT Loss
- Marrow suppression by uremia
- Reduced RBC survival span
- Dietary deficiency
- Cause related
- – – other CBC abnormalities
- Neutrophilia high ESR - in vasculitis
- Lymphopenia ESR CRP - in SLE
- Anemia in Myeloma
- Schistocytes in TMA

2. Management of Anemia

- Hb checked (yearly in CKD3, 6 monthly in 4 and 3 monthly in 5)

- First check and optimize iron levels.
- Give erythropoietin to target of 10-11.5 gm hb.
- Blood transfusion should be avoided as much as possible (Risk of fluid overload, and if transplant needed in future, risk of developing antibodies to alloantigens of transfusion will cause problems).

3. Erythropoietin

- for CKD and Chemotherapy anemia
- improves anemia thus exercise tolerance, but doesn't improve renal function
- First confirm iron levels.
- Ferritin above 100 in predialysis and above 200 in dialysis patients.
- Transferrin saturation above 25 oral iron is mostly ineffective so *IV iron is given*
- *SIDE EFFECTS*
- HTN
- Hyperkalemia
- PRCA (erythropoietin antibodies react against endogenous EPO, risk reduced by using Darbepoietin)
- Thrombosis
- Thrombocytosis
- osteoporosis
- flu like
- hypersensitivity
- iron deficiency due to increase RBC production *EPO Failure*
- inadequate dosing
- Iron deficiency
- infections
- Hyperparathyroid bone disease
- Aluminum toxicity

ELECTROLYTES

SODIUM

- dilutional hyponatremia may occur if pt continue drinking alot and output is low.
- from stage 4 onward, sodium load is poorly tolerated
- Restrict dietary sodium to less than 2gm/day

POTASSIUM (HYPERKALEMIA)

- particularly in oliguric pt
- Dietary
- ACE or ARBs

- Hypoaldosteronism
- Tissue breakdown
- (Ca-gluconate, Insulin with dextrose, Salbutamol, NAHCO3, Potassium binding resins, Dialysis)

CALCIUM

- decreased intestinal absorption due to decreased vit D
- Corrected Calcium be maintained in lower range - 8.5 to 9.5
- if *Hypercalcemia*, then

1. Switch calcium containing Ph binders to non calcium binders
2. Reduce or stop Vit D
3. Use low Calcium in dialysate

- if *hypocalcemia*, give calcium if symptomatic and particularly if PTH is also high.

PHOSPHATE

- Decreased renal excretion
- Hyperphosphatemia
- this leads to secondary Hyperparathyroidism which normalize calcium and phosphorus at expense of bones
- level may actually fall if pt is on dialysis
- Keep between 3.5 to 5.5
- Treat according to Ca-Ph product to keep it below 55.
- If product high, use non calcium binders (sevelamer and lanthanum)
- if product low, use calcium containing Binders (Calcium acetate, carbonate)

METABOLIC ACIDOSIS (HIGH ANION GAP)

- in CKD, kidneys tend to retain H°
- acidosis then cause Osteopenia, secondary Hyperparathyroidism, and protein catabolism
- treated by NaHCO3 to target of 23
- Rickets and osteomalacia
- *– – (Acidosis - high H+ - Calcium and Phosphate released from bones 5o buffer H+ - Hypercalciuria and hyperphosphaturia)*

SECONDARY HYPERPARATHYROIDISM

due to

- High Phosphate
- Low Calcium
- low vit D
- Skeletal resistance to PTH
- low FGF-23 (that is needed to keep phosphate low)

BONE DISEASE

DD

- Bone pains, fractures - Adynamic
- Prodimal muscle, axial skeleton - aluminum (Osteomalacia)
- Adynamic - likely to have low PTH, high calcium but that can't be deposited onto bone - that calcium deposits in vessels - high risk of stroke, CVS problems
- Renal osteodystrophy (high Phosphate - secondary Hyperparathyroidism - bone resorption) - high Phosphate treated by phosphate binders
- Adynamic Bone disease

RENAL OSTEODYSTROPHY

- high turnover (osteitis cystica, Primary Hyperparathyroidism)
- Low turnover (Adynamic, Metal induced osteomalacia)
- once it sets in, complete recovery is possible only by transplant

HYPERPARATHYROIDISM

1) PRIMARY

- *HIGH PTH + HIGH CALCIUM*
- By adenoma (don't respond to calcium feedback) or Hyperplasia (increase in number of cells)
- High PTH with High Calcium has few causes (primary Hyperparathyroidism, tertiary, lithium induced)
- Serum ionized calcium, albumin and intact PTH levels are checked.
- 24H urinary Calcium is measured to rule out FHH.

2) SECONDARY

HIGH PTH + LOW CALCIUM

- High Phosphate (increase PTH mRNA, decrease Calcium and calcitriol – all increase PTH)
- Low Calcium
- Low Calcitriol (because calcitriol is required to suppress PTH release)
- Low FGF-23 (FGF-23 keeps phosphate low - when FGF-23 is low, phosphate is not kept low - leads to high PTH)
- Skeletal resistance to PTH

3) TERTIARY

- Autonomous PTH secretion , mostly seen in pt of CKD/Dialysis with long standing secondary Hyperparathyroidism.
- resulting in hypercalcemia.

1) OSTEITIS CYSTICA

- key role by
- secondary Hyperparathyroidism
- along with IL 1, 6, TNF-Alpha

2) OSTEOMALACIA

- key role by heavy metals
- mainly aluminum (others are iroj, cadmium)
- Dysfunction of both osteoblasts and osteoclasts.
- Defeft in bone mineralization and bone matrix is in excess.

4) ADYNAMIC

- by PTH suppression
- no mineralization, no matrix
- – CAUSED BY

1. Aggressive supplementation of Calcium and Vit D - suppress PTH
2. Continuous Ambulatory Peritoneal dialysis
3. DM (low insulin and high RBS - suppress PTH)

- – *LABS*

1. PTH (low in Adynamic and osteomalacia, high in osteitis cystica)
2. Vit D level
3. bone specific ALP (marker of osteoclast activity - high in osteitis, low in adynamic)

4. only confirmatory test is *BONE BIOPSY*
5. Xray - subperiosteal and endosteal resorption.

- – *MANAGEMENT*

1. Vit D - if level lower than 20 units, give 20,000 iu/day vit D2 (in high turnover)
2. Low Turnover - never vit d in adynamic bone disease - vit d will increase calcium but because PTH is low, calcium won't get deposited onto bone and instead will get deposit in vascular sites
3. Cinacalcet - in secondary Hyperparathyroidism - increases calcium sensing by PTH receptors
4. Calcium - level is kept in upper normal range to keep PTH suppressed
5. Phosphate

- Level to be kept below 5.5
- Dietary
- Non Calcium Binders - *sevelamer , lanthanum* are preferred if calcium level is normal
- Calcium containing Binders *Calcium acetate, carbonate* in case of concomitant hypocalcemia

6. Acidosis correction by bucarb
7. Parathyroidectomy (subtotal)
8. If aluminum suspected - chelation with desfuroxime

- – √ *(in low turnover - PTH suppression is prevented by keeping calcium and vit d low and non calcium binders for Phosphate)*

HYPERTENSION

- CKD and ACR above 70 - 130/80
- CKD and DM - 130/80
- CKD and ACR below 70 and no DM - 140/90
- ACE inhibitors
- High dose Frusemide

FLUID

- Usually fluid overload (causing edema, pulmonary edema, HTN)
- Tubular pathology patients may have volume depletion. Tubules lose concentrating ability and result in polyuria. so patients may have low eGFR but still maintaining normal output.

GIT COMPLICATIONS

- Nausea, vomiting
- Uremic gastritis
- GIT Bleed
- Ascites
- elevated amylase (lipase is not elevated- so for suspected pancreatitis in patient of CKD - lipase is better test

CARDIAC COMPLICATIONS

- HTN (as a consequence of volume expansion, and all patients may not have edema but still have volume expansion that will cause HTN - so before starting antihypertensive its better to give diuretics and increase their dose.
- pericarditis
- fluid overload
- atherosclerosis

RESPIRATORY COMPLICATIONS

- Pulmonary Edema
- Pleural Effusion

CNS COMPLICATIONS

- Restlessness
- Encephalopathy
- seizures

ENDOCRINE COMPLICATIONS

- High PTH (secondary Hyperparathyroidism due to high Phosphate because phosphate excretion by kidneys is reduced)

AKI

- Criterion

- most recent and commonly used is KDIGO (kidney disease, improving global outcome)

1. Creatinine increase of 0.3mg in 48 hrs
2. creatinine increase 1.5times in 7 days
3. Urine volume less than 0.5ml/kg/hr for last 6 hours. (in 60kg man, less than 30ml/hour or less than 200ml in 6 hrs)

- ***Staging***

1. creat increase 1.5-1.9 of baseline
2. Creat increase 2-2.9 of baseline
3. Creat increase 3 fold, or creat above 4, or output less than 0.3ml/kg/hr

SCREENING

- All admitted pt - check vitals
- assess for AKI risk (Comorbidities - DM, CLD, PVD, HTN, Drugs)

CAUSES

- (also important is to rule out AKI on background of CKD)

PRE RENAL (60%)

- *– tubular damage is main feature –*
- (inflammatory changes, breaks in GBM, interstitial edema, sometimes Microvascular changes) - dead tubular cells may shed into lumen causing obstruction
- normally, renal autoregulatory mechanisms keep flow in normal range by afferent dilation and efferent vasoconstriction. If volume depletion becomes severe, autoregulatory mechanisms cant compensate further.

1. Hypotension
2. Volume depletion (fluid or blood loss)
3. systemic failure (liver, cardiac, sepsis) - reduction in renal perfusion despite normal volume

RENAL (30-40%)

- ATN - (exogenous nephrotoxins - aminoglycosides, Acyclovir, Cephalosporins, Contrast, Chemotherapy, Tacrolimus), Endogenous

nephrotoxins (myoglobin, hemoglobin, Paraproteins, uric acid for example in tumour lysis)

1. AIN - (Drugs beta-lactam antibiotics, penicillins, NSAIDs, PPIs, 5-ASA, infections, autoimmune conditions)

- For treatment, may give steroids, particularly within first 2 weeks

2. GN - proteinuria, hematuria, s/s of underlying cause (GBM disease, IgA, HSP, Post infective, SLE, Cryoglobulinemia, B, C, HIV, Hematological malignancies)
3. Vasculitis
4. intra renal depositions (Paraprotein in Myeloma, Myoglobin in rhabdomyolysis, Hemoglobin in Hemolytic anemia or massive transfusions, Uric Acid in tumour lysis)

- Rhabdomyolysis - CK levels upto 10-15 thousands (rhabdomyolysis causes ATN)

POST RENAL (5%)

- Obstruction (BPH, Stones, procedure, Abdominal mass, Retroperitoneal fibrosis)
- – Look for distended bladder, perform imaging

MANAGEMENT

- give fluid or blood with monitoring for overload
- – √ if large volumes needed then *Crystalloids - plabolyte, hemaccel, ringer lacter* better than Saline.
- – √ Large volume of saline will cause *hyperchloremic acidosis*
- critical pt may need inotrope
- During polyuric phase, may need supplement for Bicarb, potassium, Calcium, Phosphate, magnesium.

CKD

- presence of kidney damage (imaging, biopsy, sediments, albuminuria)
- GFR below 60 persistent for 3 months
- Patient be tested 3 times in 3 months and atleast 2 results abnormal

RISK FACTORS

- Age
- Male
- HTN (systemic HTN - glomerular capillary HTN - glomerulosclerosis)
- *– Night BP and ABPM are better indicator. In causing CKD, Systolic BP contributes more than diastolic BP –*
- Proteinuria (Reasonable indicator but not absolutely reliable)
- *– RAAS blockage improves proteinuria –*
- Metabolic factors (obesity, smoking, dyslipidemia, hyperuricemia, Insulin resistance)

SCREENING

-(recommended by KDOQI - kidney diaease outcome quality index)

- HTN, DM, above 65 age
- By Urine complete, ACR, Creatinine and eGFR

GFR DECLINE RATE

- 1ml/year in elderly with lifelong exposure to CVS risk factors (HTN, lipid, DM)
- 10ml/year in DNP
- more than 10 in acquired GN with significant proteinuria and slower decline if low albuminuria.
- ADPKD may have even faster decline
- The kidney maintains GFR, despite the progressive destruction of nephrons because the remaining normal nephrons develop hyperfiltration and compensatory hypertrophy. This helps but eventually contributes towards CKD because increased capillary pressure eventually damage capillaries and causing FSGS and later global glomerulosclerosis.

ETIOLOGY

RENAL

1. ATHEROSCLEROSIS – HTN, DM, LIPIDS, claudication, TIA
2. GN (post infectious, CLD, sickle) - fever with sore throat, recurrent hematuria, Hep B,C, maligancies, amyloidosis
3. AIN, Pyelonephritis and Contrast and reflux
4. Rheumatology
5. Vasculitides (HSP, PAN and PULMORENAL)
6. Family history (cysts in kidneys, hearing impairment)

7. Myeloma
8. Drugs

PRE RENAL

- Fluid loss, CCF CLD

POST RENAL

- Stones, dribbling, retention, urgency, hesitancy, poor stream
- Gout uric acid

CKD SHORT CASE

- GPE becomes most important part.
- – ✓ while approaching the patient, quickly assess for paleness, sallow/dark complexion wasting, double lumen.
- Nails for Leukonychia, koilonychia, half nails, finger prick marks for diabetes.
- scratch marks.
- Scars of previous double lumen.
- look and feel for Fistulas.
- Nephrectomy or renal transplant scar.
- Auscultate precordium for pericarditis and Chest for fluid overload/effusion.
- Limbs Proximal myopathy, Peripheral neuropathy.
- – ✓ (In case of renal transplant scar, look for gum hypertrophy, tremors)

FISTULA

- Educate the patient at stage 3
- – ✓ *(Look, Feel at junction, press vein tract proximal to junction, lift hand above head, Auscultate)*

1) INSPECTION

- redness
- thin shiny skin (alarming finding)
- Dilated veins in limb

- Cyanosis/Edema of hand/Arm

2) PALPATION

- – ✓ *(Three key components to be assessed. junction, inflow, outflow)*
- Place hand/fingers/palm on junction.. feel for thrill and pulsation (Thrill is maximum at junction, and some pulsations at junction is normal. hyperpulsatile or water hammer pulsations at junction are alarming finding and means stenosis of fistula. wrt thrill , Absence of thrill mean junction isn't working)
- Press vein tract proximal to junction, thrill should increase and junction should become hyperpulsatile (this means artery tract is fine)
- Lift the hand above heart, junction size should shrink

3) AUSCULTATE

- High pitch bruit is normal finding.

INTERPRETATIONS

1. *fistula with bruit over it but no needling*

- Means patient is being prepared for future need of dialysis

2. *Fistula without bruit*

- Non functional

3. *Fistula with needling*

- Needle marks stays for 2-4 days, needling means fistula was used in last 3-4 days.

4. *Iliac fossa scar* for renal transplant
5. *Posterolateral scar* for nephrectomy
6. Site of Fistulas

- Wrist radio-cephalic
- Elbow Brachio-cephalic
- Axilla baso-basallic

7. approach of double lumen

- right Internal jugular
- left internal jugular

- right subclavian
- femoral